



NOTES

INFECTIONS & INFLAMMATION OF THE HEART

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Heart infections, inflammation (may affect epicardium, myocardium, endocardium)
- **May include:** infective endocarditis, Libman-Sacks endocarditis, myocarditis, rheumatic fever
- May cause/be caused by/coexist with other infections

COMPLICATIONS

- Heart failure, arrhythmias, fibrosis
- Infective, Libman-Sacks endocarditis can cause
 - **Damage to heart valves:** dysrhythmias, valve dysfunction
 - **Invasion of myocardium:** heart failure, heart block, sepsis
 - **Vegetation can embolize to extremities:** infarction/ischemia causing stroke, pulmonary edema, glomerulonephritis

SIGNS & SYMPTOMS

- See individual disorders

DIAGNOSIS

- See individual disorders

TREATMENT

- See individual disorders

VISIT...

LANZAROTE
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INFECTIVE ENDOCARDITIS

osms.it/endocarditis

PATHOLOGY & CAUSES

- Infection of endocardium, usually with bacteria, may include heart valve
- Valves have small blood vessels → damage to valve, vessels → microbes in blood escape into valvular tissue/microbes enter small vessels → infection
- Valve endothelial lining damaged
- Microbes enter body via: dental/surgical procedures, injection with infected needle/infected substance, wound/abscess
- Vegetation: fibrin, leukocytes, microbes attach to thrombosis → abnormal growth → potential embolism
- Often affects left side heart valves
 - Predisposing conditions: mitral valve prolapse, bicuspid aortic valves

TYPES

- Classified by microbial cause
 - Acute bacterial endocarditis: infection of normal valves, rapid progression
 - Subacute bacterial endocarditis: indolent infection of abnormal valves (e.g. *S. viridans*)
 - Endocarditis in IV drug users: Methicillin-resistant *Staphylococcus aureus* (MRSA), *Pseudomonas*, *Candida*
 - Prosthetic valve endocarditis: *Staphylococcus epidermidis* within 60 days of replacement; after 60 days, resembles native valve endocarditis

CAUSES

Viridans streptococci (most common)

- Low virulence
- Found in mouth
- Attacks previously damaged valves
- Small vegetations: don't destroy valve

Staphylococcus aureus

- High virulence

- Found on skin
- Infects damaged, healthy valves
- Large vegetations: can destroy valve
- Most commonly contracted from IV drug use

Staphylococcus epidermidis

- Infects prosthetic material (e.g. prosthetic heart valves)
- Enters body during valve surgery/infected IV catheter: sticks around valve/catheter
- Nosocomial infection (infection in hospital)
- Gut flora
 - *Enterococcus faecalis*
 - *Streptococcus bovis*
- Severe colorectal disease (e.g. colorectal cancer/ulcerative colitis): bacteria migrate into bloodstream

Coxiella burnetii

- Exposure to infected animals (e.g. cows, sheep, goats)
- Q fever → months/years later, endocarditis
- Affects those at high risk: immunocompromised, pregnant individuals, pre-existing heart valve defect
- Diagnosis difficult

Candida albicans

- Fungal endocarditis
- Connected with IV drug use

Culture-negative endocarditis

- Cannot be linked to bacteria using blood cultures
- Aortic vascular infection, persistent low fever, rash
- Often caused by *Coxiella burnetii*

HACEK organisms

- *Haemophilus*, *Aggregatibacter*, *Cardiobacterium*, *Eikenella*, *Kingella*
- Gram-negative bacteria
- Normal flora of mouth, throat

Nonbacterial thrombotic endocarditis

- Damage in valve exposes collagen, tissue factor → platelets, fibrin adhere → form tiny thrombosis → mitral valve regurgitation
 - Bacteremia → bacterial attach to thrombi → bacterial endocarditis

RISK FACTORS

- Valvular problems
 - Mitral valve prolapse
 - Bicuspid aortic valves
 - Prosthetic valves
 - **Valves affected:** mitral > aortic, tricuspid
- Congenital cardiac defects
- Damage to valves due to rheumatic heart disease
- IV drug use (esp. tricuspid valve)
- Chronic hemodialysis
- Poor dentition

SIGNS & SYMPTOMS

- Anorexia, weight loss, fatigue
- See mnemonic below

**MNEMONIC: FROM JANE****Signs & Symptoms****F**ever**R**oth spots: antigen-antibody complex deposits in eyes**O**sler nodes: painful antigen-antibody complex deposits in pads of digits**M**urmur: turbulent blood flow past damaged heart valve**J**aneway lesions: erythematous lesions due to emboli; small, painless, flat**A**nemia**N**ail-bed hemorrhage (splinter hemorrhages): deposition of emboli**E**mboli: vegetations detach from valve, deposit elsewhere (nail beds, kidneys, spleen, central nervous system)**DIAGNOSIS****DIAGNOSTIC IMAGING****Chest X-ray**

- Enlarged heart, possible pulmonary congestion

Echocardiogram

- Inflamed heart muscle walls, dilation

LAB RESULTS

- **Elevated troponin, creatine kinase levels** (due to heart muscle damage)

Cardiac muscle biopsy

- Definitive diagnosis
- Risky procedure, performed only if test results would change treatment plan

OTHER DIAGNOSTICS**ECG**

- Sinus tachycardia (increased heart rate)
- T-wave inversions
- "Saddle-shaped" ST segment elevations

TREATMENT

- **Viral:** improves slowly over time
- Arrhythmias resolve as inflammation improves

MEDICATIONS

- Antibiotics
- **Signs of heart failure:** managed with medication, fluid balance

SURGERY

- Heart transplant in severe cases (e.g. Chagas, giant cell myocarditis)



Figure 12.1 Janeway lesions are hemorrhagic macules or nodules that may appear on the palms of the hands or soles of the feet in cases of infective endocarditis.

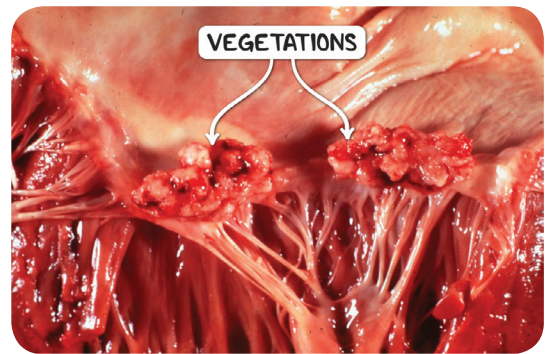


Figure 12.2 Bacterial vegetations on the mitral valve in endocarditis.

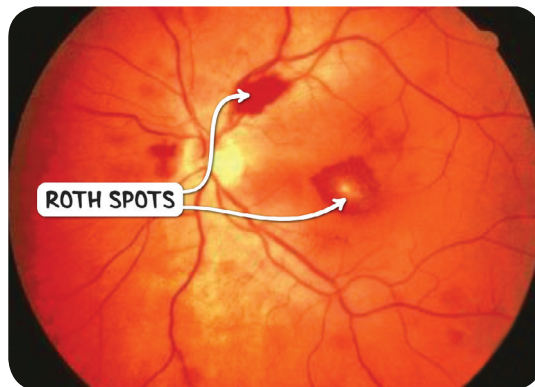


Figure 12.3 Roth spots seen in the retina.

LIBMAN–SACKS ENDOCARDITIS

osms.it/endocarditis

PATHOLOGY & CAUSES

- Autoimmune endocarditis associated with systemic lupus erythematosus (SLE), advanced malignancy, rheumatoid arthritis
- AKA nonbacterial thrombotic endocarditis/verrucous endocarditis

CAUSES

- Antigen-antibody complexes settle in endocardium
 - Arises on valves /chordae tendineae, most often mitral valve
 - Arises even on atrial/ventricular endocardium
 - Sterile vegetations: aortic valves

COMPLICATIONS

- Damage to heart valves
- Invasion of myocardium
- Vegetations may embolize
- In rare cases, may cause secondary infective endocarditis

SIGNS & SYMPTOMS

- Regurgitant murmurs
 - Bilateral vegetations on valve leaflets
- Clinical manifestations indicate systemic emboli
 - Kidney: flank pain, hematuria
 - Skin: rash, digital ischemia
 - Cardiac/central nervous system (CNS): chest pain, stroke

DIAGNOSIS

- Must exclude infective endocarditis (may coexist)

DIAGNOSTIC IMAGING

Transesophageal echocardiogram (TEE)

- Small, warty, vegetations on both atrial and ventricular sides of valves
- Regurgitation, valve insufficiency

LAB RESULTS

- CRP, WBC levels, and antiphospholipid/anticardiolipin antibody level may aid in differentiation

TREATMENT

- Treat underlying SLE

MEDICATIONS

Anticoagulants

- E.g. heparin, direct thrombin, Xa inhibitors
- Address embolic risk

MYOCARDITIS

osms.it/myocarditis

PATHOLOGY & CAUSES

- Inflammation of/damage to myocardium
- Swelling impairs myocardial contraction → less blood pumped out of heart with each heartbeat

CAUSES

Coxsackieviruses A & B infections

- Viral infections → lymphocytic myocarditis: B, T cells, water invade interstitial space
- Common in North America

Trypanosoma cruzii

- Single-cell protozoan → Chagas disease
- Amastigotes within heart muscle cells (intracellular stage of trypanosomes) → necrosis of heart muscle cells
- Common in South America

Trichinella

- Intestinal roundworm may move into heart → myocarditis

Borrelia burgdorferi

- Lyme disease bacterium

Toxoplasma gondii

- Single cell parasite harbored by cats

Systemic lupus erythematosus (SLE)

- Non-infectious myocarditis
- Immune system attacks myocardium

Drug-associated/hypersensitivity

- Adverse drug reaction inflames heart
- Eosinophils enters blood vessels in myocarditis

Giant cell

- Inflammation of heart from unknown cause
- Macrophages fuse to form single giant cell

RISK FACTORS

- Viruses that cause flu-like illnesses, HIV/AIDS, Lyme disease, strep, staph infections, parasites

COMPLICATIONS

- Heart failure, fibrosis, arrhythmias

SIGNS & SYMPTOMS

- Clinical manifestations of heart failure (e.g. fatigue, shortness of breath, hepatomegaly, edema)
- Acute heart failure → cardiogenic shock
- Arrhythmias (e.g. ventricular fibrillation, ventricular tachycardia) → sudden cardiac death
- Fever
- Positional chest pain, related to pericarditis: better/worse depending on body's position

DIAGNOSIS

DIAGNOSTIC IMAGING

Chest X-ray

- Enlarged heart, possible pulmonary congestion

Echocardiogram

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LAB RESULTS

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OTHER DIAGNOSTICS

ECG

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- “Saddle-shaped” ST segment elevations



MNEMONIC: BCD ST3G

Common Causes of Myocarditis

Borrelia burgdorferi

Coxsackieviruses A and B

Drug-associated

Systemic lupus erythematosus

Trypanosoma cruzi

Trichinella

Toxoplasma gondii

Giant cell

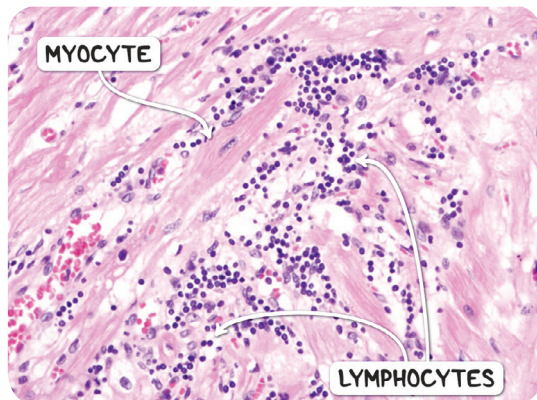


Figure 12.4 Histological appearance of myocardium in viral myocarditis.

TREATMENT

- **Viral:** improves slowly over time
- Arrhythmias resolve as inflammation improves

MEDICATIONS

- Antibiotics
- **Signs of heart failure:** managed with medication, fluid balance

SURGERY

- Heart transplant in severe cases (e.g. Chagas, giant cell myocarditis)

RHEUMATIC FEVER

osms.it/rheumatic-heart-disease

PATHOLOGY & CAUSES

- Autoimmune inflammatory disease caused by complication of streptococcal infection
- Develops after streptococcal pharyngitis (strep throat) from Group A beta hemolytic streptococcus

CAUSES

Molecular mimicry

- Antibodies against streptococcal M-protein cross-reacts with proteins on myocardium, heart valves, joints, skin, brain → cytokine-mediated inflammatory response
- Inflammation results in widespread pathology

Pancarditis

- Inflammation of endometrium, myometrium, pericardium (three layers of heart tissue)
- Myometrium: Aschoff bodies (microscopically viewed nodules caused by inflammation) → leads to fibroid necrosis
 - Characteristic feature of pancarditis
 - Anitschkow cells (enlarged macrophages inside Aschoff bodies), caterpillar-like nuclei
- Pericardium: pericarditis causes pain, friction rub due to visceral pericardium rubbing against parietal pericardium

Chronic rheumatic heart disease

- Repeated exposure to group A beta-hemolytic streptococcus → immune attacks on tissues (esp. heart tissue)
- Valves (typically mitral valve, sometimes aortic) develop scar tissue → leaflets thicken, fuse → commissural fusion
 - Stenosis AKA “fish-mouth”/“buttonhole” stenosis
 - Regurgitation (blood flows backward)
- Chordae tendineae attached to valves thicken

TYPES

- When only a subset of symptoms present, classified as the following

Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections (PANDAS)

- Neuropsychiatric symptoms

Poststreptococcal reactive arthritis

- Joint symptoms

RISK FACTORS

- Small number of individuals with strep throat develop rheumatic fever, more likely in children/those in areas of poverty, crowding
- Rheumatic fever primarily affects children 5–7 years old, 20 days after infection
- One third of cases asymptomatic

SIGNS & SYMPTOMS

Acute rheumatic fever

- Following symptoms develop 2–4 weeks after streptococcal pharyngitis
- Fever
- Migratory polyarthritis of joints: temporary inflammation, swelling, joint pain
- Erythema marginatum: non-itchy, reddish rash, rings on arms/trunk
- Subcutaneous nodules: firm collagen lumps under skin
 - Reaction to hypersensitivity
 - Painless
 - Back of wrist, outside elbow, front of knee
- Pancarditis (inflammation of three layers of heart)
- Dyspnea, sharp chest pain
- Friction rub heard on auscultation due to pericarditis
- Impaired ability of heart to contract

(myocarditis) → heart failure, death

- **Sydenham's chorea:** rapid, jerky movements of face, arms from damage to basal ganglia
 - Autoimmune reaction on basal ganglia of brain
 - Appears late (three months after infection)

Chronic rheumatic heart disease

- **Symptoms dependent on type of damage to heart:** aortic stenosis, aortic regurgitation, mitral stenosis, mitral regurgitation, pulmonic regurgitation

PANDAS

- Pediatric, abrupt onset, episodic course of symptoms
- **Neurologic abnormalities:** motoric hyperactivity (fidgeting), choreiform movements in stressed postures (sudden, jerky movements), frank chorea (rapid, irregular, jerks, movements continuous while awake but improve with sleep)
- Obsessive-compulsive disorder/tic disorder

Poststreptococcal reactive arthritis

- Arthritis occurring after a streptococcal infection

DIAGNOSIS

OTHER DIAGNOSTICS

Jones criteria for acute rheumatic fever

- Evidence of previous group A streptococcus infection plus two major criteria/one major plus two minor criteria



MNEMONIC: JONES

Major criteria

Joints: polyarthritis
myOcarditis: O = vaguely heart-shaped

Nodules: subcutaneous

Erythema marginatum

Sydenham's chorea

- Minor criteria
 - **Signs/symptoms:** fever ($>38.5^{\circ}\text{C}/101.3^{\circ}\text{F}$), arthralgia
 - **Laboratory evidence:** increased acute phase reactants ($\uparrow$ erythrocyte sedimentation rate, $\uparrow$ C-reactive protein, $\uparrow$ leukocytosis)
 - **Electrocardiograph:** prolonged PR interval
- Evidence of recent infection
 - **Positive** throat culture
 - **Positive** rapid antigen detection test
 - **Elevated** antistreptolysin O titre (ASO)
- **Exception:** Sydenham's chorea/pancarditis independently may indicate rheumatic fever
- Electrocardiogram changes

Chronic rheumatic heart disease

- Previous repeated cases of rheumatic fever
- **Diagnosis depends on damage done to heart:** aortic stenosis, aortic regurgitation, mitral stenosis, mitral regurgitation, pulmonic regurgitation



Figure 12.5 Anitschkow cells (enlarged macrophages with linear nucleoli) in an Aschoff body (a granuloma) in a case of rheumatic myocarditis.

TREATMENT

MEDICATIONS

Rheumatic fever

- **Goals of treatment:** control, eradicate streptococcus, prevent complications, relieve joint pain, relieve fever
 - **Antibiotics:** penicillin G
 - **Anti-inflammatory agents:** aspirin, non-steroidal anti-inflammatory drugs (NSAIDs), steroids
 - **Antipyretics:** NSAIDs
 - Rest

OTHER INTERVENTIONS

Rheumatic fever

- Maintain dental health
- **Strict long-term, prophylaxis:** history of bacterial endocarditis, heart transplant, artificial heart valve, other congenital defect

Rheumatic heart disease

- Prevent repeated attacks/acute rheumatic fever, streptococcal infections
- **History of acute rheumatic fever:** prophylactic treatment for extended period (benzathine penicillin G/oral penicillin V, 10 years to life)

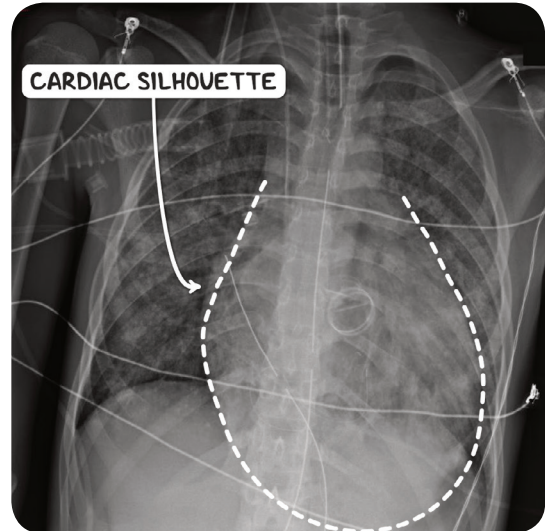


Figure 12.6 Massive cardiomegaly secondary to aortic and mitral valve disease in a severe case of rheumatic fever.

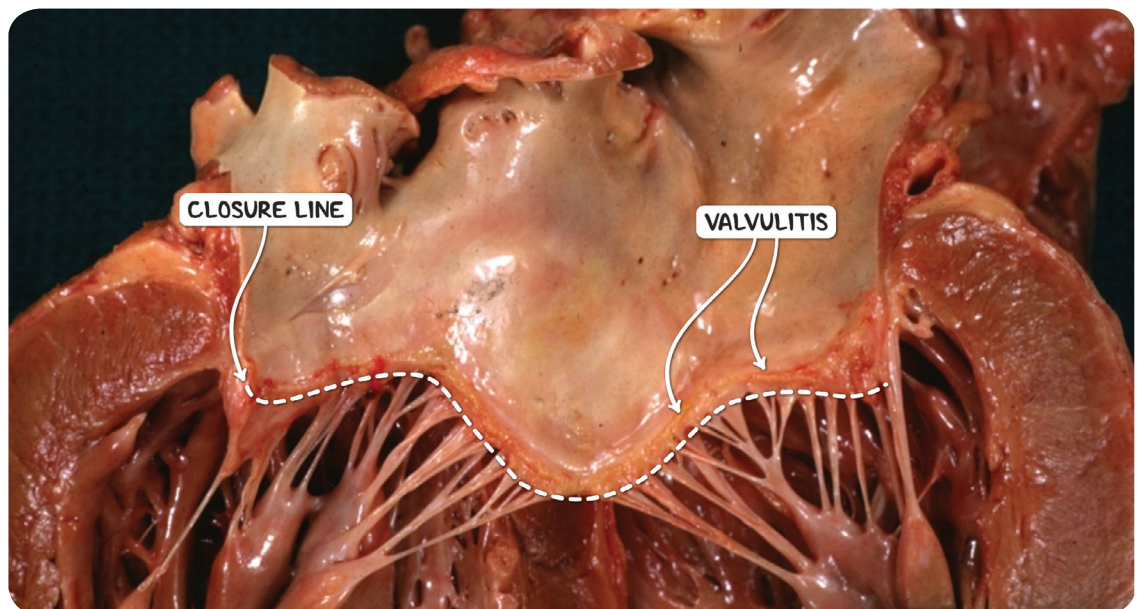


Figure 12.7 Gross pathology of acute rheumatic endocarditis; there is a line of acute inflammation (valvulitis) along the closure line of the mitral valve.